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Evidence from prospective cohort studies does not support current dietary fat guidelines: a systematic review and meta-analysis

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Abstract (248 words)

Objectives: National dietary guidelines were introduced in 1977 and 1983, by the United States (US) and United Kingdom (UK) governments to reduce coronary heart disease (CHD) mortality by reducing dietary fat intake. Our 2016 systematic review examined the epidemiological evidence available to the dietary committees at the time; we found no support for the recommendations to restrict dietary fat. The present investigation extends our work by re-examining the totality of epidemiological evidence currently available relating to dietary fat guidelines.

Methods: A systematic review and meta-analysis of prospective cohort studies currently available, which examined the relationship between dietary fat, serum cholesterol and the development of CHD, were undertaken.

Results: Across 7 studies, involving 89,801 participants (94% male), there were 2,024 deaths from CHD during the mean follow-up of 11.9 ± 5.6 years. The death rate from CHD was 2.25%. Eight data sets were suitable for inclusion in meta-analysis; all excluded participants with previous heart disease.

Risk ratios (RR) from meta-analysis were not statistically significant for CHD deaths and total or saturated fat consumption. The risk ratio (RR) from meta-analysis for total fat intake and CHD deaths was 1.04 (95% CI 0.98 to 1.10). The RR from meta-analysis for saturated fat intake and CHD deaths was 1.08 (95% CI 0.94 to 1.25).

Conclusions: Epidemiological evidence to date found no significant difference in CHD mortality and total fat or saturated fat intake and thus does not support the present dietary fat guidelines. The evidence *per se* lacks generalisability for population-wide guidelines.

Introduction

US public health dietary advice was announced by the Select Committee on Nutrition and Human needs in 1977[1] and was followed by UK public health dietary advice issued by the National Advisory Committee on Nutritional Education in 1983.[2] Dietary recommendations in both cases focused on reducing dietary fat intake; specifically to i) reduce overall fat consumption to 30% of total energy intake and ii) reduce saturated fat consumption to 10% of total energy intake.

The recommendations were intended to address mortality from coronary heart disease (CHD). In 2015, we published a systematic review and meta-analysis,[3] which reported that evidence from randomised controlled trials (RCTs), available to the dietary guideline committees, did not support the introduced dietary fat recommendations. In 2016, we published a systematic review and meta-analysis,[4] which reported that RCT evidence currently available does not support the introduced dietary guidelines. In 2016, we published a systematic review,[5] which reported that epidemiological evidence, available to the dietary guideline committees, did not support the introduced dietary fat recommendations. The aim of these systematic reviews has been twofold: to examine the epidemiological and RCT evidence base for the dietary fat guidelines to assess if they were justified at the time of their introduction and to review if the evidence currently available supports the extant recommendations. This systematic review and meta-analysis completes this work by examining the totality of epidemiological evidence currently available.

Methods

This systematic review and meta-analysis uses the Meta-analysis Of Observational Studies in Epidemiology methodology (MOOSE).[6] MOOSE uses parts of the Cochrane methodology for systematic review and meta-analysis of RCTs, Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA), for example the figure for presenting search methodology.[7] To examine best practice evidence available, this review has focused on prospective cohort studies.[8]

Search strategy

A search was undertaken to identify prospective cohort studies that examined the relationship between dietary fat intake, serum cholesterol and mortality from CHD. Exclusion criteria were: clinical trials; cross-sectional studies; case control studies. Inclusion criteria were: prospective cohort studies; participants were human adults; primary study outcome was CHD mortality; data related to dietary fat consumption were available; data on CHD mortality and serum cholesterol measurements were available.

The search was undertaken on 30 September 2015. No date limitations were set, enabling the earliest possible papers to be included. Searches of the literature were performed using MEDLINE, Embase and the Cochrane Library (Fig. 1).

Selection of studies

Of 669 identified articles, 607 were rejected upon review of the title and abstract. Of these, 55 were rejected for being review articles. 55 were commentaries, editorials or letters. 39 were clinical trials, 9 were cross sectional or case control studies. 75 related to conditions other than heart disease, primarily cancer, diabetes, hypertension and obesity. There were 53 studies in which animals or children/adolescents were the primary focus. 98 were articles about pharmacology/blood analysis. 123 were rejected for being related to a particular food, nutrient or supplement, rather than dietary fat. A further 40 papers were educational material and 36 reviewed dietary compliance in nutritional studies. 24 articles were mathematical modelling exercises, including meta-analysis. 62 papers remained. 13 were rejected on closer inspection of the full paper: 12 were reviews, commentaries or expert opinion pieces and 1 was educational material. Where an abstract was unavailable, the publication type, journal name and meta-tags were reviewed to assess if the article should be rejected (for example “letter”, “clinical trial”, “paediatric”). Copies of the remaining articles were

obtained from university libraries or the British Library. 49 papers, *prima facie*, met the inclusion criteria.

Of the 49 papers included in qualitative synthesis, 18 were found *prima facie* to meet the inclusion criteria and 31 were duplications of these 18 studies. On detailed examination of the 18 studies, 6 were the studies included in Harcombe *et al*, [5] which were available to the UK National Advisory Committee in 1983. The Western Electric study; [9 10] The Puerto Rico Heart Health Program; [11-13] The Seven Countries Study; [14] The Framingham Heart Study; [11 15] The Honolulu Heart Program; [11 16 17] and a study conducted in London and the South East. [18] None of these had data conducive to meta-analysis. 24 papers were additional publications related to these 6 studies; the majority of these were about The Seven Countries Study. The additional publications were examined for data published after the introduction of the dietary fat guidelines to assess if any Harcombe *et al* [5] studies could be updated. A 1984 paper about The Honolulu Heart Program [19] and a 1991 paper about The Framingham Heart Study [20] were reviewed, but neither reported CHD mortality.

A total of 12 new studies were found. [21-32] Seven papers were additional publications related to these studies. Four studies were excluded on closer inspection for not reporting CHD mortality data [22 23 25 26] and another one contained CHD mortality data that were not separable from CHD events. [24] These five studies also failed to meet inclusion criteria for other reasons: no saturated fat data; [22 26] no total fat data; [25 26] no serum cholesterol data. [22-24 26] Mann *et al* was the only study to review animal fat, not all dietary fat. [26] This study, examining the benefits of a vegetarian diet, reported consumption of total animal fat and saturated animal fat, thus excluding the fat and saturated fat in many non-animal foods. [33]

Six studies did not report serum cholesterol data, [21 27 28 30-32] which left one study meeting the full inclusion criteria. [29] The serum cholesterol component part of the diet-heart hypothesis was set aside as an inclusion criterion, which enabled data for dietary fat (total and saturated) and CHD mortality to be examined for seven studies. [21 27-32] The UK Health survey [21] presented data for men and women separately and The Strong Heart Study of American Indians [31] presented data for those aged 47-59 years and those aged 60-79 separately. Table 1 presents the extracted data for total and saturated dietary fat and CHD deaths for these seven studies; nine information sets.

To ascertain the validity of eligible observational studies, a pair of reviewers (ZH and BD) worked independently to determine which studies met the inclusion criteria. The same one additional study was agreed upon as meeting the original diet-heart hypothesis criteria and the same seven studies were agreed upon for the revised search. One of the authors (ZH) examined contemporary systematic reviews of prospective cohort studies [34-36] to ensure that no studies had been omitted or included inconsistently. Correspondence was entered into with one author for thorough verification. [37] Risk of bias was assessed using the Cochrane Collaboration assessment tool for component parts relevant to observational studies, defined as follows: selection bias (cohort appropriately reflected wider population characteristics); detection bias (blinding of outcome assessment); attrition bias (incomplete data outcome); and reporting bias (selective reporting) (Fig. 2). [38]

Data Extraction

Table 1 details data extraction of: study name; year of key publications; participant characteristics; whether free from CHD at study entry; years of follow-up; CHD deaths; outcomes relating to total dietary fat and saturated dietary fat for CHD deaths.

Statistical Analysis

The overall pooled effect was calculated using random effects meta-analysis. This takes into account between study variance when assigning weights in meta-analysis. The weighting of studies is more balanced with random effects than fixed effects methodology; larger studies have less impact and smaller studies have more impact than with fixed effects. Heterogeneity and bias were

quantified using the I^2 and T^2 calculations, $I^2 = 100\% \times (Q - df)/Q$, where Q is Cochran's heterogeneity statistic and df the degrees of freedom. Funnel plot methodology and Egger's regression intercept[39 40] were calculated to assess risk of publication bias. Analyses were performed using Comprehensive Meta-Analysis.[41]

Meta-analysis was possible for all but one study in Table 1.[27] Kushi *et al* reported fat intake as a percentage of overall energy intake for those who died from CHD and those who did not. However, no standard deviations or standard errors were presented, which are necessary for inclusion in meta-analysis. Forest plots for the associations between CHD deaths and total fat and saturated fat are presented in Fig. 3 and Fig. 4 respectively.

Fig. 1 Summary of systematic review profile[7].

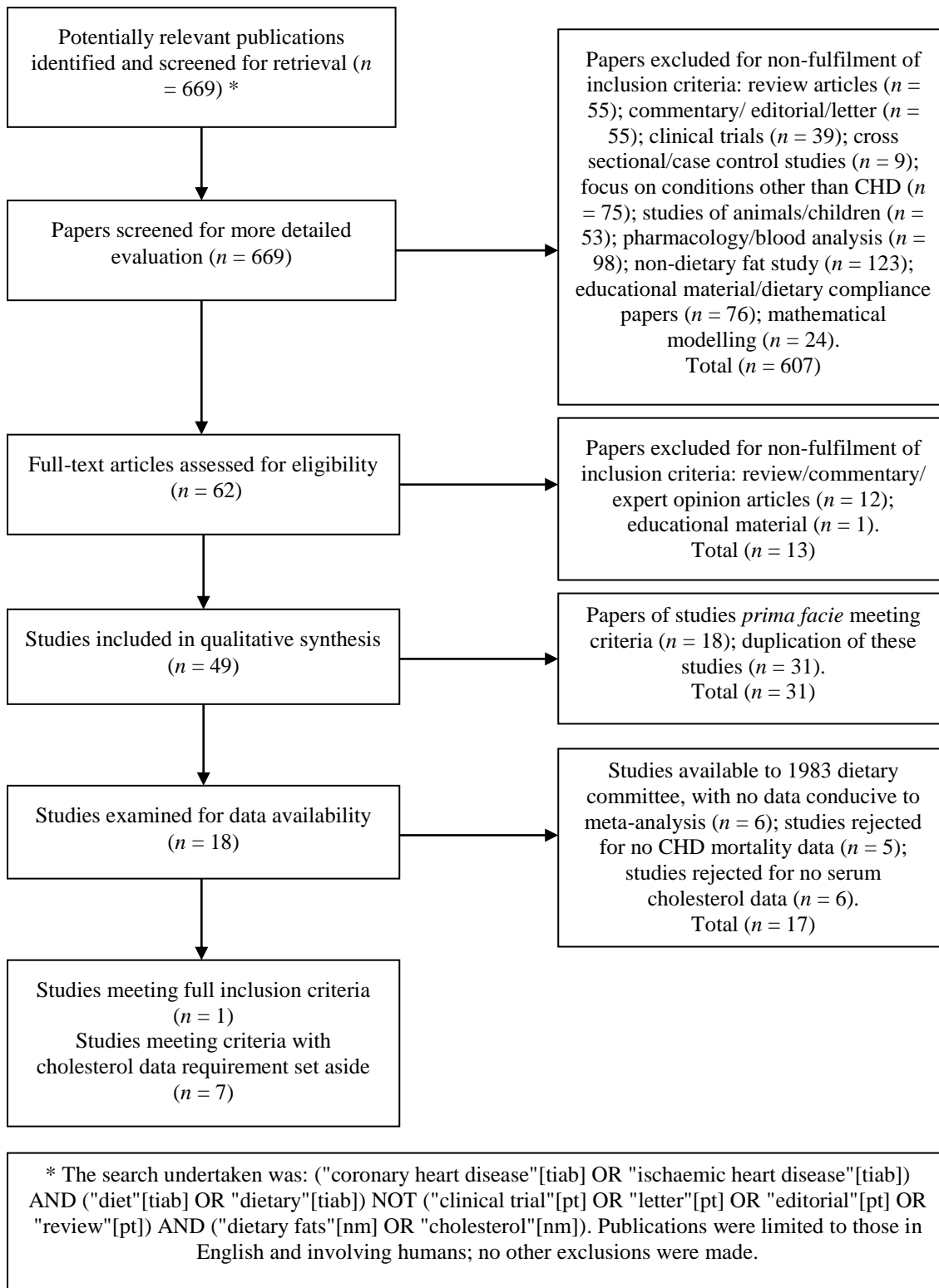


Table 1 Outcome data from prospective cohort studies for: study name; participant number, gender and age range; years of follow-up; total fat and saturated fat for CHD deaths vs. CHD-free.

Study	Men/Women /Age	CHD- free?	Follow- up yrs	CHD deaths	Total fat CHD death/Non	Sat fat CHD death/Non
Kushi <i>et al</i> , Ireland Boston study[27] (1985)	1,001 M (30-69)	N	20	110	39.4/38.5 % energy (*)	17.4/16.9 % energy (*)
Ascherio <i>et al</i> , US Health Professionals[28] (1996)	43,757 M (40-75)	Y	6	229	Note 1 1.59 (1.01 - 2.51) (<i>p</i> =0.02)	Note 1 2.21 (1.38 - 3.54) (<i>p</i> =0.0016)
Esrey <i>et al</i> , Lipid Research [29] (1996)	2,071 M 1,854 W (30-59)	Y	12	52	Note 2a 1.04 (1.01 - 1.08) (<i>p</i> <0.01)	Note 2a 1.11 (1.04 - 1.18) (<i>p</i> <0.01)
	282 M 339 W (60-79)	Y	12	40	Note 2a 1.00 (0.96 - 1.04) (<i>p</i> <0.01) (*)	Note 2a 0.97 (0.89-1.05) (<i>p</i> <0.01) (*)
Pietinen <i>et al</i> , The Finnish Cancer Study [30] (1997)	21,930 M (50-69)	Y (all smokers)	6.1	635	Note 1 0.85 (0.65 - 1.12) (*)	Note 1 0.73 (0.56 - 0.95) (<i>p</i> = 0.044)
Boniface & Tefft, UK Health Survey[21] (2002)	1,225 M (40-75)	Y	16	98	Note 2b 1.01 (0.93 - 1.10) (*)	Note 2b 1.00 (0.86 - 1.18) (*)
	1,451 W (40-75)	Y	16	57	Note 2b 1.19 (1.03 - 1.37) (<i>p</i> = 0.0181)	Note 2b 1.40 (1.09 - 1.79) (<i>p</i> = 0.0074)
Xu <i>et al</i> , Strong Heart Study[31] (2006)	646 M 1,013 W (47-59)	Y	7.2	46	Note 3 3.57 (1.21 - 10.49) (<i>p</i> = 0.01)	Note 3 5.17 (1.64 - 16.36) (<i>p</i> = 0.01)
	405 M 874 W (60-79)	Y	7.2	92	Note 3 0.77 (0.41 - 1.45) (*)	Note 3 0.80 (0.41 - 1.54) (*)
Nagata <i>et al</i> , Japanese Study[32] (2012)	12,953 M (≥35)	Y	16	665	Notes 1 & 4 1.12 (0.80 - 1.57) (*)	Notes 1 & 4 0.96 (0.67 - 1.39) (*)
TOTAL	89,801			2,024		

Table notes: (*) Not statistically significant
































95% confidence intervals in parentheses.

Note 1: Data were presented in quintiles. The lowest quintile fat intake was given a risk ratio (RR) for CHD mortality of 1.0. The RR for the highest fat intake, quintile 5, was presented.

Note 2: 2a) This presented the RR for a one unit increase in the percentage of energy intake provided by the nutrient.[29] 2b) This was defined as an additional 100 grams per week.[21]

Note 3: Data were presented in quartiles. The lowest quartile fat intake was given a risk ratio (RR) for CHD mortality of 1.0. The RR for the highest fat intake, quartile 4, was presented.

Note 4: Data are for cardiovascular disease (CVD), not CHD. Nagata *et al* included 15,403 women, but did not report on CVD, or CHD mortality by fat intake for women (table 4, p.1718 for men)[32].

Key				
	Low risk of bias			
	High risk of bias			
	Unclear risk of bias			
	Cohort appropriately reflected wider population (selection bias)			
	Blinding of outcome assessment (detection bias)			
	Incomplete outcome data (attrition bias)			
	Selective reporting (reporting bias)			
Ireland Boston/Kushi (1985)				
US Health Professionals/Ascherio (1996)				
Lipid Research/Esrey (1996)				
Finnish Cancer Study/Pietinen (1997)				
UK Health Survey/Boniface (2002)				
Strong Heart Study/Xu (2006)				
Japanese Study/Nagata (2012)				

Participants were followed for a minimum of 6 years and a maximum of 20 years. The mean duration of the seven cohorts was 11.9 ± 5.6 years. The weighted mean duration (person years by participants) was 8.3 ± 1.1 years.

All trials had complete outcome data, avoiding attrition bias (Fig. 2).[38] One study was judged unclear for selection bias, as participants excluded non-smokers.[30] Four studies were judged unclear for detection bias, as blinding of outcome assessment was unclear.[27 30-32] Risk of reporting bias was judged low for four of the studies: it was judged unclear for The Strong Heart Study and The Lipid Research Study, as the rationale for the age group divisions were not clear;[29 31] and it was judged high risk for the Japanese Study as CVD by fat intake was researched, but not reported, for women.[32] (Fig. 2)

For total fat and CHD deaths, Esrey *et al* carried the greatest weight, 31.76% for the 30 to 59 age group and 29.45% for the 60 to 79 age group (Fig. 3 random effects methodology).[29] The two Boniface and Tefft observations, for men and women, carried a combined weight of 30.04%.[21] The two Xu *et al* observations carried a weight of 1.02% between them.[31] The risk ratio for the meta-analysis of the eight data sets available for total fat and CHD deaths was 1.04 (95% CI 0.98 to 1.10). The overall effect measurement lies on the line of no effect. There was no statistically significant difference between total fat and CHD deaths.

The meta-analysis for total fat and CHD deaths was tested for sensitivity analysis of the exclusion of any one study. There were no circumstances in which the exclusion of any one study made the overall effect size significant.

There was evidence for between study heterogeneity. The Q-value was 18.218 (8 *df*) and this was statistically significant $p = 0.020$. I^2 was 56.087 and T^2 was 0.002 indicating difference in true effects.

Visual inspection of the funnel plot revealed that one study was outside the standard error funnel for the meta-analysis of total fat and CHD deaths. This was the Xu *et al* study for people aged 47-59,[31] which was substantially different from other risk ratios. The Egger's regression test indicated no statistically significant asymmetry for total fat and CHD deaths. The Egger's regression intercept was 0.640 (95% CI, two-tailed, -0.959 to 2.238), but this was not statistically significant (one-tailed $p = 0.188$; two-tailed $p = 0.376$).

For saturated fat and CHD deaths, Esrey *et al* carried the greatest weight, 19.83% for the 30 to 59 age group and 19.28% for the 60 to 79 age group (Fig. 4 random effects methodology).[29] The two Boniface and Tefft observations, for men and women, carried a combined weight of 28.96%.[21] The two Xu *et al* observations carried a weight of 5.10% between them.[31] The risk ratio for the meta-analysis of the eight data sets available for saturated fat and CHD deaths was 1.08 (95% CI 0.94 to 1.25). The overall effect measurement lies on the line of no effect. There was no statistically significant difference between saturated fat and CHD deaths.

The meta-analysis for saturated fat and CHD deaths was tested for sensitivity analysis of the exclusion of any one study. There were no circumstances in which the exclusion of any one study made the overall effect size significant.

There was evidence for between study heterogeneity. The Q-value was 37.080 (8 *df*) and this was statistically significant $p < 0.001$. I^2 was 78.425 and T^2 was 0.025 indicating difference in true effects.

Visual inspection of the funnel plot revealed that three studies were outside the standard error funnel for the meta-analysis of total fat and CHD deaths[28 30] and the Xu *et al* study for people aged 47-59.[31] The Egger's regression test indicated no statistically significant asymmetry for saturated fat and CHD deaths. The Egger's regression intercept was 0.686 (95% CI, two-tailed, -1.978 to 3.351), but this was not statistically significant (one-tailed $p = 0.281$; two-tailed $p = 0.562$).

Fig. 3 Estimates of CHD mortality and total fat risk ratios (95% confidence intervals) from meta-analysis.

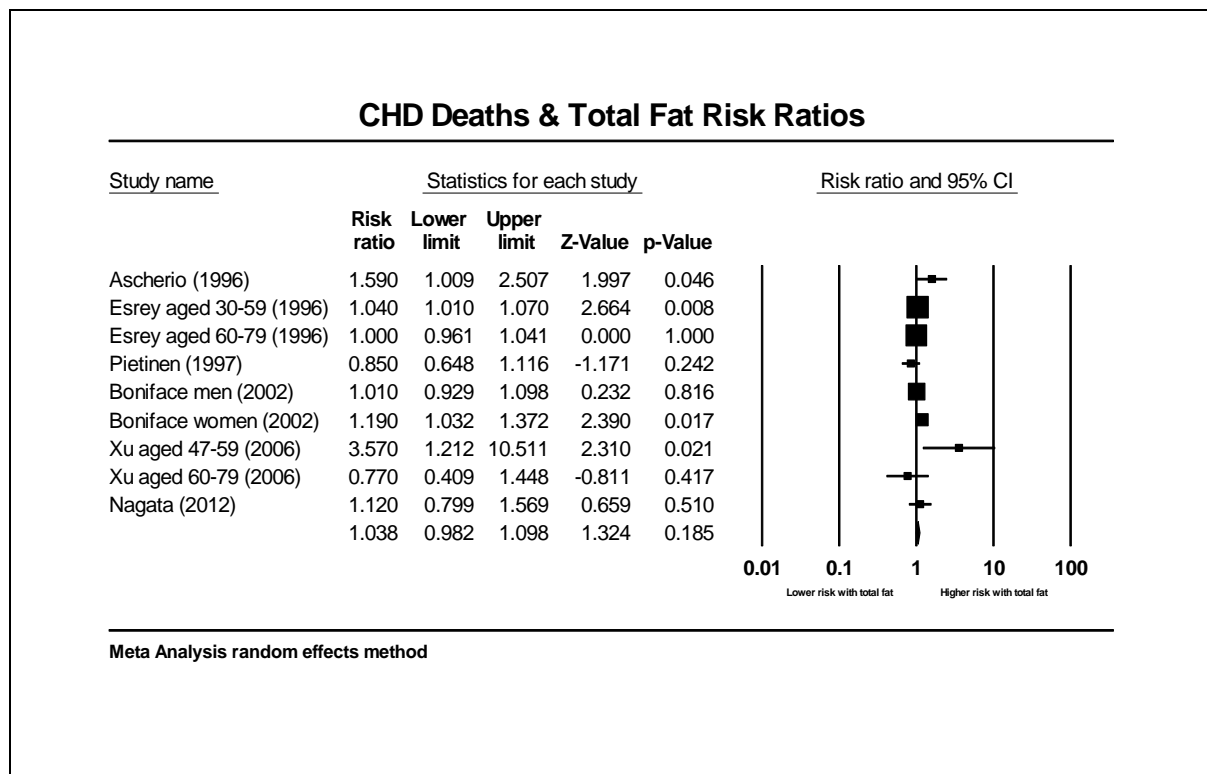
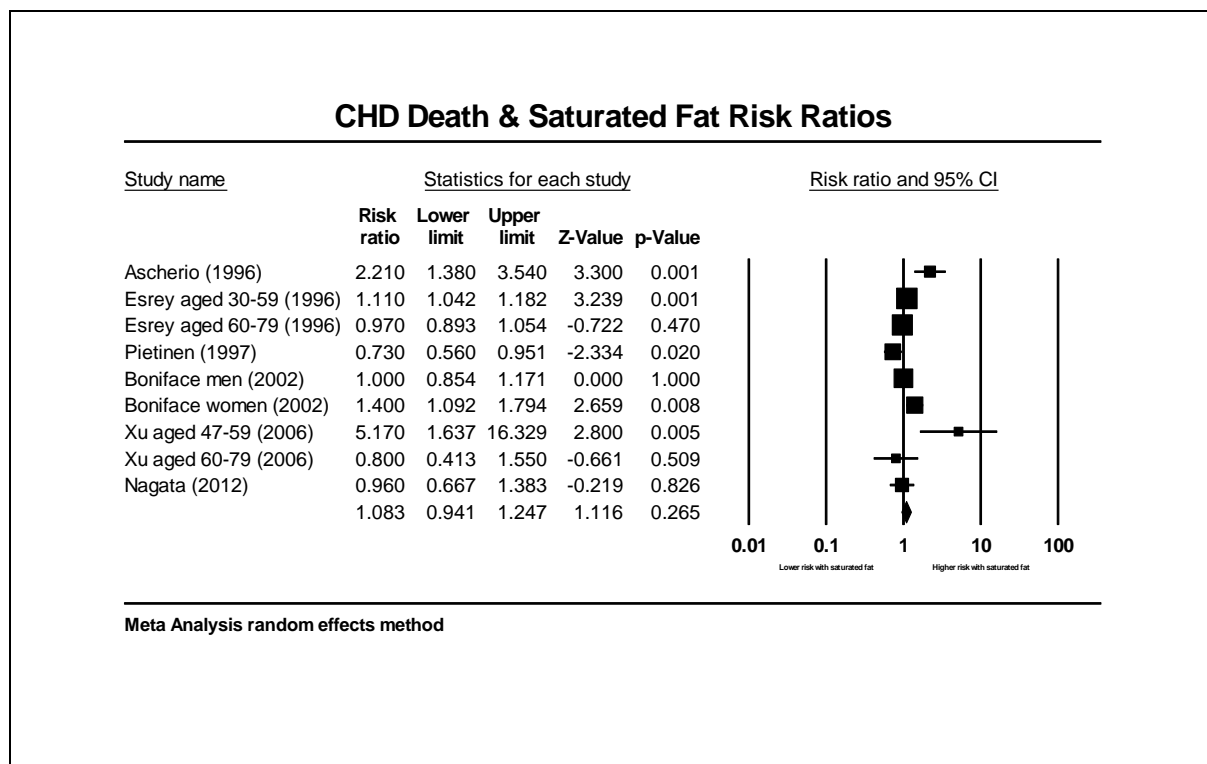


Fig. 4 Estimates of CHD mortality and saturated fat risk ratios (95% confidence intervals) from meta-analysis.



Examination of the dietary guidelines

None of the seven cohort studies examined either of the introduced dietary guidelines: a total fat consumption of 30%, or a saturated fat consumption of 10%, of energy intake. Two studies examined the total fat intake and the saturated fat intake, as a percentage of calorie intake, for participants who died from CHD compared with those who did not.[27 29] Esrey *et al* also reported the risk ratio for an additional one unit of total or saturated fat as a percentage of energy intake. Three studies measured total and saturated fat intake in quintiles and reported data for CHD mortality as risk ratios comparing the lowest and highest quintiles of intake.[28 30 32] The Strong Heart Study followed this method, but for quartiles, not quintiles.[31] The remaining study reported the risk ratio for an additional 100 grams of total or saturated fat per week for men and women separately.[21]

CHD mortality

Across 7 studies, involving 89,801 participants, there were 2,024 deaths from CHD during the period of follow-up. The death rate from CHD was 2.25%, during the mean follow-up of 11.9 ± 5.6 years. The death rate was influenced by the large Finnish study, which included only participants who smoked. The death rate for this study was 2.9%.

The death rate contrasts with the 30% death rate for the six RCTs reviewed in Harcombe *et al*[3], which reinforced the high death rate in secondary studies. The one study that included men with previous heart disease reported a death rate of 11%.[27]

Significance reported by the studies

Three of the seven studies reported some participants separately: two by age groups[29 31] and one by gender.[21] This enabled 10 sets of data to be examined (nine in meta-analysis) (Table 1). Six found no significant relationship between total fat and CHD mortality,[27 30 32] men in the UK health and lifestyle survey[21] men and women aged 60 to 79 years in The Lipid Research Study[29] and the participants aged 60 to 79 years from The Strong Heart Study.[31] The remaining four found a significant relationship between total fat and CHD mortality: Ascherio *et al*:[28] women in the UK health and lifestyle survey;[21] and the < 60 year old participants from The Lipid Research and The Strong Heart Study.[29 31] Five found no significant relationship between saturated fat and CHD mortality;[27 32] men in the UK health and lifestyle survey;[21] and the participants aged 60 to 79 years from The Lipid Research and The Strong Heart Study.[29 31] The remaining five found a significant relationship between saturated fat and CHD mortality;[28-30] women in the UK health and lifestyle survey;[21] and the under 60 year old participants in The Lipid Research and The Strong Heart Study.[29 31]

Meta-analysis confirmed that the pooled data were not significant for CHD mortality and total fat intake or CHD mortality and saturated fat intake.

Serum cholesterol levels

The only study to meet the full systematic review inclusion criteria was Esrey *et al*.[29] This study found a significant relationship between baseline serum cholesterol and subsequent mortality from CHD in the age group 30 to 59 years, but not in the age group 60-79 years old.

Discussion

The main findings of this systematic review and meta-analysis are that the epidemiological evidence currently available to the dietary committees provides no statistically significant retrospective support for the introduction of dietary fat guidelines.

Most studies reported other significant findings: Kushi *et al* found a significant association with higher intake of carbohydrate and fiber and lower CHD mortality. Esrey *et al* found significant positive associations with age, male gender, Body Mass Index (BMI), smoking and CHD deaths. Two studies reported significant associations with trans fat intake and CHD mortality.[28 30]

Pietinen *et al* additionally reported significant associations with the number of cigarettes smoked per day, the number of years of smoking, blood pressure, BMI and CHD mortality. Pietinen *et al* found that High Density Lipoprotein (HDL) cholesterol, education and physical activity were inversely related to the risk of CHD. Ascherio *et al* found that associations found between dietary fat and heart disease were greatly attenuated by adjusting for fibre intake. Boniface and Tefft reported that smoking, not exercising and being socially disadvantaged were related to higher saturated fat intake and CHD death, which meant that confounding variables could have played a part in findings. Xu *et al* found that age, being male, having diabetes and smoking were all significantly associated with CHD deaths.

Design limitations

As was found with the review of RCT evidence in Harcombe *et al* [3 4], the fundamental design limitation of epidemiological evidence currently available is that it lacks generalisability. RCT evidence available to the dietary guideline committees had studied men with pre-existing heart disease; [3] epidemiological evidence available to the dietary guideline committees had studied men mostly free from heart disease. [5] One RCT currently available included men and women, without previous heart disease, and contained all component parts of the diet-heart hypothesis for examination. [42] One prospective cohort study currently available included men and women free from heart disease and contained all component parts of the diet-heart hypothesis for examination. [29]

The method of extracting dietary information was a limitation. Dietary recall is generally unreliable and 24-hour recall may not be representative of usual diet. [43 44] Dietary surveys, where food is weighed at the time of being recorded, are also unreliable. [45 46]

Study conclusions

Two studies concluded that evidence was strong for an association between dietary fat and death from CHD. Boniface and Tefft reported: “Strong evidence was found for the within cohort relationship of dietary fat and CHD death in women while no evidence was found for a relationship in men” (p.786). [21] Xu *et al* found that: “Total fat, saturated fatty acid, and monounsaturated fatty acid intake were strong predictors of CHD mortality in American Indians aged 47-59 y” (p.894). [31]

The Japanese Study found: “Total fat intake is associated with decreased mortality in Japanese men but not in women” (p.1713). [32]

Two studies reported weak or no associations between dietary fat and CHD mortality. Ascherio *et al* appeared to reference The Seven Countries Study with their comment, “These data do not support the strong association between intake of saturated fat and risk of coronary heart disease suggested by international comparisons” (p.84). [28] Pietinen *et al* reported that “There was a significant positive association between the intake of trans fatty acids and the risk of coronary death. There was no association between intakes of saturated or cis-monounsaturated fatty acids or linoleic or linolenic acid or dietary cholesterol and the risk of coronary death” (p.876). [30]

The final two studies described the relationship as unclear. Kushi *et al* concluded: “The nature of the association in this study between dietary lipids and the risk of mortality from coronary heart disease remains unclear” (p.816). [27] The one study meeting all systematic review inclusion criteria reported: “We conclude that future research must be directed toward better understanding the pathway between dietary intake and coronary disease as the current diet-lipid-heart hypothesis may be overly simplistic” (p.211). [29]

Other meta-analyses of prospective cohort studies

The aim of this systematic review, as part of a series of four, [3-5] has been to evaluate the RCT and prospective cohort evidence base for two specific dietary fat guidelines (30% and 10% of energy

intake in the form of total and saturated fat respectively) at their time of introduction and the present day. This has not previously been undertaken.

A number of other systematic reviews and meta-analyses have been undertaken, which are relevant to the present review.[34-36 47] De Souza *et al*[47] systematically reviewed saturated, not total, fat including any observational study, not just prospective cohort studies, with mortality as the end point. Siri-Tarino *et al*[34] undertook a meta-analysis of prospective cohort studies for saturated, not total, fat, with disease as the end point. Chowdhury *et al*[36] also examined saturated, not total, fat and with disease, not mortality, as the end point, including RCTs and prospective cohort studies in the same review. Skeaff and Miller[35] were closest to this review, and the one reviewing RCTs currently available,[4] having undertaken a systematic review and meta-analysis of RCTs and prospective cohort studies combined, for total and saturated fat, with CHD end points, disease and mortality. This was published in 2009.

De Souza *et al* concluded: “Saturated fats are not associated with all cause mortality, CVD, CHD, ischemic stroke, or type 2 diabetes, but the evidence is heterogeneous with methodological limitations” (p.1).[47]

The conclusion of Siri-Tarino *et al*’s review was “meta-analysis of prospective epidemiologic studies showed that there is no significant evidence for concluding that dietary saturated fat is associated with an increased risk of CHD or CVD” (p.535).[34]

Chowdhury *et al* set out to summarise evidence between fatty acids and coronary disease. Their review examined saturated, monounsaturated, polyunsaturated and trans fats, while also reviewing individual chain length fatty acids, palmitic (C16:0) and margaric (C17:0) as examples. The conclusion was “Current evidence does not clearly support cardiovascular guidelines that encourage high consumption of polyunsaturated fatty acids and low consumption of total saturated fats” (p.398) [36].

Skeaff and Miller sought to summarise the evidence from cohort studies and RCTs of the relation between dietary fat and risk of CHD. Their conclusion was “Intake of total fat was not significantly associated with CHD mortality. Intake of total fat was also unrelated to CHD events” (p.175) [35].

Harcombe *et al*[3 5] found that the dietary fat guidelines were not supported by RCT or epidemiological evidence available at the time of their introduction. These dietary fat guidelines have prevailed until 2016 and thus the validity of their evidence base remains important to examine. UK dietary fat guidelines are unchanged. The Dietary Guidelines for Americans, issued in January 2016,[48] were conspicuously silent on the subject of total fat, but reiterated that saturated fat should be restricted to no more than 10% of calorie intake. Harcombe *et al*[4] found that the RCT evidence currently available does not support the dietary fat guidelines. This review finds that the epidemiological evidence currently available does not support the dietary fat guidelines. All systematic reviews found serious limitations with the availability of primary prevention, both-sex, studies, which are the ones most likely to have generalisability for whole populations.

The conclusion of the four systematic reviews and three meta-analyses is that there was no evidence to support the dietary fat guidelines being introduced and there is no evidence currently available to support them. Public health authorities need to urgently review dietary advice.

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